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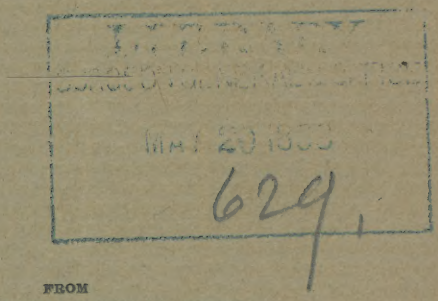

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SEPTIC PERFORATION OF THE RIGHT INTERNAL CAROTID ARTERY.

Autopsy Five Months After Death.[1]

BY A. JACOBI, M.D., AND JAMES EWING, M.D.,
of New York.

THE clinical history of the case, which was learned five months after death, began two weeks before death with ordinary symptoms of croupous tonsillitis; there were membranes on both tonsils. The report of the New York Health Department gave no Klebs-Loeffler bacilli. After one week the throat was pronounced clear and the child was much better. Monday, May 2d, about one week after the beginning of the illness, the patient had a severe chill, with rise of temperature, pain in the throat, and dysphagia. The lymph-nodes on both sides of the neck soon became much swollen. The temperature rose considerably and the patient was much prostrated. On May 4th there was a considerable hemorrhage from the nares and pharynx. On May 6th a second very profuse hemorrhage occurred, "filling a bowl" with apparently arterial blood. Its color was described as being bright. On May 8th there was a third severe hemorrhage. The nares were then plugged. On May 10th, early in the afternoon, the plugging was removed from the nares while the child was struggling much, and a fourth severe and fatal hemorrhage occurred, blood coming from the nose and mouth. The exact point of issue of the previous hemorrhages was not ascertained.

The autopsy was held 5 months and 8 days after death, on October 18th. The body had been interred in dry gravel, in a wooden casket, surrounded by a casing-box of pine. The outer box was quite dry. The casket was moist, with no molds seen inside or out. Running from the foot of the casket was a blood-stained line which had slightly soiled the covering of the coffin and the bottom of the box. The internal coverings were moist, the clothing of the cadaver much decayed in places, especially over and under the trunk. Covering the clothing and the skin of the entire body was a layer of coarse, brownish dust, in which were many small white insects, the size of a pin-head, and their small oval eggs. This

[1] Paper read and specimen exhibited before the Association of American Physicians at Washington, D. C., May 5, 1898.

dust, composed of the insects and excreta, had produced a dark-brown discoloration of the skin, especially of the face, which was nearly black in places. The body was rigid, the skin much desiccated and over the fingers almost horny. The nails and hair were firm, the teeth very loose. The orbits contained a little black, grumous fluid; the lids and lashes were intact. The cheeks, chin, nose, and features were apparently natural and not shrunken.

The tissues of the neck were more moist than elsewhere. Over the right parotid, the sternomastoid, and in the posterior temporal region there was marked œdema of all the tissues. The lymph-nodes of both sides of the neck were much swollen, down to the clavicles. The tissues of the posterior pharyngeal wall were very dark but not necrotic, especially on the right side. A large cavity, filled with black fluid in one compartment and bright blood in the deeper portions, was found bounded internally by the right tonsil and pharyngeal wall, which here was very thin. It reached about 2 cm. below the tonsil, above nearly or quite to the bony vault of the pharynx, and it extended posteriorly to the deep tissues of the neck and involved the internal carotid and its branches in their irregular ramifications. This cavity consisted of one older, smooth-walled portion lying nearest the tonsil and just behind the thinned pharyngeal wall, and measured about 2 cm. in diameter. The rest of the cavity appeared to be of more recent formation, and was filled with fresh blood, partly clotted. The internal carotid, 1.5 cm. above its origin, showed an irregular opening 5 mm. in circumference, freely communicating with the deeper portion of the above cavity. On the posterior wall of the pharynx, at the level of the tip of the epiglottis, was a ragged opening of similar dimensions, leading into the older and superficial portion of the cavity. The tonsils showed numerous deep excavations, but no ulcerations. The mucous membrane of the nares was in an advanced state of decomposition, the periosteum was loose, the vomer free from its articulations. There were no evidences of hemorrhage in this region. The larynx appeared quite normal, but was filled with reddish-brown fluid. The trachea was normal.

The lungs were very firm, the pleura desiccated and shiny. There were 4 oz. of yellowish, transparent fluid in the pleural cavities. The lungs showed numerous lobules sharply marked off, dark reddish, filled with blood. In the right lower lobe one area was softened, gelatinous, decolorized, resembling clots found in some veins. The lungs otherwise were very anæmic.

The heart was very hard and tightly contracted. The right side contained a firm, dark clot of moderate size. The left was entirely empty. The muscle, though anæmic, was normal. Its valves were normal.

The liver was much desiccated superficially and very firm,

very light leaden in color. The markings were indistinguishable. Section showed numerous small cavities, some of which are patent bloodvessels. The gall-bladder was contracted and empty. The spleen was moderately enlarged, the capsule smooth, hard, and dry; it was dark gray on section. The Malpighian bodies were light gray and very distinct.

The kidneys were normal in size, very firm in consistence, and dry. On section, they were very light-colored and anæmic. The pyramids were moderately congested, the markings distinct and regular. The adrenals were slightly desiccated. The pancreas was very firm, dry, and appeared normal. The œsophagus was contracted and very hard.

The stomach was tightly contracted, firm, its mucous membrane well preserved, and thrown into prominent rugæ. It contained a little semifluid matter, of the color of coffee-grounds. The peritoneum was shiny, very dry, and light-colored, like paper. An abnormal band constricted the ascending colon about its middle, and the cecum appeared considerably dilated.

The colon contained considerable brownish desiccated fecal matter. The muscular system was very dry, tough, and of a bright-red color. The aorta and large arteries were very tough, dry, and shrunken. The large veins were firm, and contained a little clotted blood; in some places they were decolorized and gelatinous.

On microscopical examination, the wall of the pharyngeal (abscess) cavity was found to be composed of intensely inflamed granulation-tissue in the older portion, and of connective and muscular tissue infiltrated with serum; blood and leukocytes were found in the deeper portion. A section of the wall of the carotid, just above the point of rupture, showed this vessel running through an area of advanced purulent inflammation. At this point, the adventitia was œdematous, the muscular coat split up, and at one point very thin. The intima appeared intact. In the same section the wall of the internal jugular vein was so infiltrated with leukocytes and serum that its structure was almost indistinguishable.

The cervical lymph-nodes showed an intense exudative inflammation, but no collections of pus were seen. They showed considerable post-mortem necrosis. The consolidated lobules of the lungs were filled with a homogeneous reddish substance, without signs of inflammation, either in the parenchyma or bronchi.

The cells of the liver were partly necrotic; the cell-bodies stained faintly, were extensively vacuolated and coarsely granular; the cell-membranes were very distinct and the nuclei faint. Many capillaries were stuffed with leukocytes, which sometimes appeared in collections of 50 to 100, both mononuclear and polynuclear.

The cells of the kidneys showed usually partial granular

fragmentation, but many appeared entirely intact and normal. There were no evidences of an inflammatory process.

In the spleen there was considerable increase of mononuclear and polynuclear cells, both in the pulp and in the Malpighian bodies. All the structures were well preserved.

The stomach-wall was normal, with very little superficial post-mortem necrosis of the epithelial lining.

Sections through the wall of the cavity, stained by methylene-blue, showed numerous colonies of cocci. No bacilli or chain-cocci could be distinguished.

The anatomical diagnosis was peritonsillar and retro-pharyngeal abscess; inflammation and rupture of the right internal carotid artery; healed croupous inflammation of the tonsils; purulent inflammation of the large branch of the internal jugular vein.

Epicrisis.—The history and pathologic examination indicate positively the development of a peritonsillar and retropharyngeal abscess following a croupous tonsillitis. As no Klebs-Loeffler bacilli were found in the bacteriologic examination of the Health Board, the croupous inflammation was of staphylococcous origin, in accordance with the above results of the bacteriological examination. The first hemorrhages must have resulted from rupture, following inflammation of veins or arteries attacked by the advancing suppuration. The fatal hemorrhage undoubtedly came through the openings demonstrated in the internal carotid artery and pharyngeal wall, a necessarily fatal lesion. The condition of the internal jugular vein indicates that it or its branches were possibly the point of origin of the first hemorrhage. The second hemorrhage, which is reported to have been very copious and of bright color, must have been arterial.

Is it possible to make a correct diagnosis of the source of such a hemorrhage? Many a case may be decided by the character of the blood, whether arterial or venous; but in many septic conditions the color of the arterial blood is no longer bright, and from a large septic cavity the discharge may be both arterial and venous. In exceptional cases (as was remarked by Guthrie 50

years ago, and by Lidell, Roux, Wahl, and others) a large lacerated artery with a ragged interior or irregular edges may, while bleeding, exhibit a murmur. In other cases, when the question is between a hemorrhage from the carotid and the nasopharynx, the digital compression of the artery is, in an urgent case, provided a medical man is present, more likely to answer the inquiry as to the source of the bleeding.

During the progress of an inflammatory and suppurative process round cells are caused to proliferate near the vasa vasorum of the media, and the endothelia of the intima multiply rapidly. In this way the resistance of the vascular tube is increased, and many a threatened calamity may thus be avoided. When, however, a rupture of the bloodvessel-wall has taken place, the hemorrhage depends on the size of the laceration, and also on the condition of the patient. For every hemorrhage creates a disposition to its cessation by lowering of the blood-pressure; there may even be syncope, and a clot may form. But within a single day, or several days, the amount of circulating blood is increased, the heart becomes more vigorous, and the clot is expelled. In cases of ulcerous erosion, when the patient has suffered from a septic process, circumstances are least favorable. In them there are rarely long intervals between the hemorrhages. Fatal cases of that nature are not very common. A certain number have been observed in syphilis, sometimes in the course of an arteritis, mainly in the young, in whom the skin, the brain, or the periosteum is frequently subject to bleeding from that cause. Metrorrhagia has been observed under the same circumstances (Bradley). Gummata located in the bloodvessel-walls, or more often in their neighborhood, will ulcerate and lacerate. Thus Bernhard (*Lancet*, 1872) had to ligate the common carotid because of formidable hemorrhages originating in the larynx. Morell

Mackenzie[2] cites the case of a hemorrhage probably from a vertebral artery, as the patient expectorated the transverse process of his epistropheus. Landrieux[3] describes the case of a patient who had gummas in very many organs, besides periostitis. His pharynx was full of ulcerations and cicatrices; one of the ulcerations communicated with the internal carotid. He died of the second hemorrhage, which took place two days after the first.

Parenchymatous or venous bleeding, more or less copious, sometimes fatal—as in a case of Rosenthal's—in scarlatina, diphtheria, and hemophilia, or in septic processes complicated with leukocythemia or scurvy, have been seen frequently. Trifling extravasations from dilated bloodvessels behind the uvula or in the nasopharynx, which now and then are mistaken for hemoptysis, I do not here count. In a single case I saw also a fatal series of hemorrhages in an old man who had inaccessible sclerotic veins in his posterior nares.

What concerns us here is a fatal hemorrhage from erosion of an artery, complicated with phlebothrombosis. The lesion of the artery was evidently the result of local suppuration. Eighty-eight such cases of hemorrhage from large arteries caused by suppuration were collected by Monod. In 37 they were occasioned by abscesses in the soft parts, viz., tonsils, retropharynx, lymph-bodies of the neck, and secondary processes in typhoid fever and in scarlatina. In many cases the internal carotid, in two the aorta were injured. Monod has also two cases of inguinal buboes with erosion of the femoral artery; Koenig, who in 1889 quoted Monod, suggested that many of these hemorrhages were caused by "pus bonum et laudabile;" in 1898 he would not make this distinction, at least not with the same assur-

[2] *Diseases of the Throat and Nose.*
[3] *Bull. Soc. Anat.*, Paris, July, 1874.

ance. For there is undoubtedly a destructive effect as well of an uncomplicated staphylococcous process as of a mixed infection.

Fatal cases of erosion of large bloodvessels in the throats of septic children are, however, not very frequent. I knew of no case when I published my "Treatise on Diphtheria" in 1880. V. Gautier, it is true, had two fatal cases of pharyngeal hemorrhage—one quoted from Rendu, who found the carotid and its branches intact—and one of his own, in which vertebral caries was found, but the source of the hemorrhage was not discovered.[4] Greenhow[5] quotes Williams, who had a fatal case, in which the patient died apparently from the profuse discharge of bloody material resembling claret from the throat, amounting to two pints a day, but here, too, the local lesion was not found. Still, Becquerel had previously published[6] a case of death resulting from an ulcerous lesion of the inferior pharyngeal artery. Quoting this case, Greenhow adds: "Probably some of the cases of fatal hemorrhage on record in this country (England) have really arisen from gangrenous ulceration." Carmichael[7] related the case of a baby of five weeks, who had a feverish cervical adenitis. Respiration and deglutition became worse in two days, and the patient died in a hemorrhage. The retro-pharyngeal abscess communicated with the post-tonsillar cavity; the open artery was the external carotid.

Bokay and Alexey have recorded[8] the case of a boy of four years, who had an inflamed throat three weeks. No diagnosis was made. Then his fever rose to 40° C., his face became œdematous his throat red, but no local ab-

[4] Des abscès retro-phar. ou de l'angine phlegmoneuse. Bale, 1869.
[5] Diphtheria, 1860, p. 209.
[6] *Gaz. Méd. de Paris*, 1843, p. 692.
[7] *Edin. Med. Jour.*, July, 1881.
[8] *Jahrb. f. Kinderheilk.*, xvii, p. 209. 1881.

scess or swelling was discovered. There being dyspnea, the case was considered one of probable pneumonia. Besides, he had a nephritis—probably scarlatinous. One night there was a hemorrhage of 350 cu. cm., and the next morning another. The autopsy was made by Szekeres, under whose name this case appears in some of the records, so that it has often been counted twice. There was pus in a tonsil and in the surrounding tissue, and in a cavity of the size of a hazel nut was the perforation of the common carotid.

The new, elaborate and exact *Traité des Maladies des Enfants*[9] has only this to say on the subject, that gangrene will sometimes advance into large blood-vessels.

[9] In 5 volumes, by Grancher, Comby & Marfan (I, p. 555).